THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

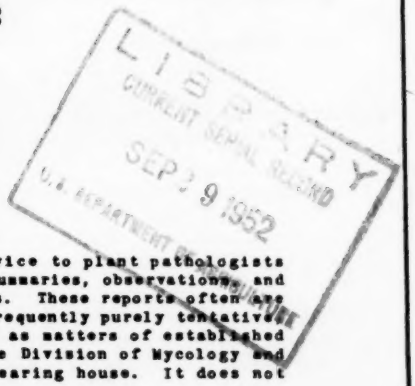
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 36 Number 9

September 15, 1952



The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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Plant Industry Station — Beltsville, Maryland
Volume 36 — September 15, 1952 — Number 9

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PLEASE NOTE: Copy for illustrations for the Reporter should be sent unmounted (see PDR 36(5): 214. May 15).

A SERIOUS OUTBREAK OF DWARF BUNT IN TALL OAT GRASS IN OREGON[1]

John R. Hardison and Malcolm E. Corden[2]

Dwarf bunt was found in the Tualatin variety of oat grass in Union County, Oregon by Mr. Ray Wendel, county agricultural agent, and Mr. Elmer Johnson, certification specialist, when inspecting grass fields for certification during June 1952. Tualatin oat grass is probably more closely related to Arrhenatherum elatius var. bulbosum (Willd.) Spenner than to the common tall oat grass, A. elatius (L.) Presl.

Tualatin oat grass plants infected with dwarf bunt were severely dwarfed as shown in Figure 1. Infected panicles are shortened and compressed, Figure 2. The difference in height between infected and healthy culms may permit combine harvesting above the diseased panicles. Careful harvesting should exclude most of the smut balls from the healthy seed. Processing the seed with specific gravity separators using air flotation will probably remove any smut balls remaining in the field-run seed.

FIGURE 1. Habit of wheat dwarf bunt in Tualatin oat grass.

FIGURE 2. Abnormal inflorescences of Tualatin oat grass caused by wheat dwarf bunt. Diseased on left. Healthy on right.

The disease was found in five fields varying from a trace to about 50 percent dwarfed culms. The heavily infested fields have a past history of severe dwarf bunt infection in wheat. Dwarf

[1]Co-operative investigations between the Division of Forage Crops and Diseases, U. S. Department of Agriculture and the Department of Botany and Plant Pathology, Oregon Agricultural Experiment Station.

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bunt on wheat was first found in Oregon in this area near Enterprise in 1948.

Examination of the greenish black smut balls reveals that the chlamydospores have the proper size and prominent reticulations characteristic for the wheat dwarf bunt formerly referred to as a race of Tilletia caries (DC.) Tul. Spores placed in water, water agar, and potato dextrose agar failed to germinate after one week. Dormancy of spores is another feature that typifies most collections of wheat dwarf bunt.[3]

Dr. George W. Fischer very kindly advised the writers that he had recently described the dwarf smut fungus as a separate species, Tilletia brevifaciens.[4] Referring to the Oregon specimens of intermediate wheatgrass, Agropyron intermedium (Host) Beauv., and Tualatin oat grass, Dr. Fischer stated in a recent letter "I have made mounts from both collections using Shear's mounting medium and unquestionably both collections are Tilletia brevifaciens. The hyaline sheath is especially distinct in the Agropyron collection."

Information on susceptibility of grasses to dwarf bunt is not available. In recent correspondence, Dr. C. S. Holton suggested the inability to reproduce the disease from seed inoculation as a fundamental reason why the grass host range of dwarf smut has not yet been determined.

Infection of Tualatin tall oat grass by the dwarf bunt organism is unusual as no bunt has been recorded on Arrhenatherum, and dwarf bunt has previously been known only on grasses in the tribe Hordeae.[4, 6] Infection by the dwarf smut fungus of a grass in the tribe Aveneae suggests that other grasses outside the tribe Hordeae may prove susceptible. Infected perennial grasses may represent important host plants for perpetuating the organism and for the development of new pathogenic races infectious to wheat in an area even though resistant varieties of wheat are being grown.

Dwarf bunt was seen also in several wheatgrasses in the same area in 1952. This is logical, since various species of Agropyron and Hordeum are known to be susceptible to races of common bunt (T. caries) by natural occurrence and artificial inoculations,[5, 6] and dwarf bunt was reported on two species of Agropyron from Idaho.[4]

Dwarf smut represents a serious threat to the seed production of several forage grasses in the Pacific Northwest and the Rocky Mountain areas where this disease occurs. Studies are in progress to determine the range of susceptibility in the economic grasses grown in this area.

To cope with this serious, new problem, the Oregon standards for grass seed certification were changed in July 1952 and now include the dwarf smut tolerances that are used in wheat certification. The maximum tolerance allowed in field inspection is 0.5 percent. There is a zero tolerance for whole smut balls in seed inspection. Grass seed from the infested areas will be cleaned separately and will be chemically treated to kill any seed-borne spores. These measures will prevent dissemination of dwarf smut in grass seeds produced in Oregon.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS, OREGON

[3]Holton, C.S., R.H. Bamberg, and R.H. Woodward. 1949. Progress in the study of dwarf bunt of winter wheat in the Pacific Northwest. Phytopath. 39: 986-1000.

[4]Fischer, George W. 1952. Tilletia brevifaciens sp. nov., causing dwarf smut of wheat and certain grasses. Research Studies of the State College of Washington 20, No. 1: 11-14.

[5]Fischer, George W. 1936. The susceptibility of certain wild grasses to Tilletia tritici and Tilletia levis. Phytopath. 26: 876-886.

[6]_____. 1945. The Ustilaginales or "Smuts" of Washington. Washington Agricultural Experiment Station, Bulletin No. 459. 84 pp.

DISTRIBUTION AND PREVALENCE OF PHYSIOLOGIC RACES OF THE LEAF RUST OF WHEAT IN THE UNITED STATES IN 1951

C. O. Johnston and M. N. Levine

The physiologic race content of 316 field collections of the leaf rust of wheat caused by Puccinia rubigo-vera tritici, made in 27 States in 1951, was determined at Manhattan, Kansas or St. Paul, Minn. Complete sets of 8 differential varieties were used at both rust laboratories in 1951. This resulted in a listing of many races of minor importance that are very similar to certain major races such as 5, 9, 15, and 126.

The results of the determinations are summarized in Table 1. Although 30 races were isolated, only 5 can be considered as abundant and widely distributed. These were races 5, 9, 15, 58, and 126. Races 2, 6, 52, and 105 also were abundant in some localities but were not so widely distributed as the preceding 5 races. Race 5, representing 27.0 percent of all isolates, was by far the most abundant. It was isolated from collections from all but 6 States. The dominance of race 5 is even more striking when it is realized that race 52, which represented 8.2 percent of all isolates, is similar to race 5 and sometimes is indistinguishable from it.

Race 126 was second in importance representing 13.6 percent of all isolates and occurring in all but 4 States. Race 105 is similar to Race 126.

One of the features worthy of special note is the increasing abundance and distribution of race 15 and the related race 2. Race 15 was third in abundance and was isolated from collections in 22 States. It is a virulent race on many wheat varieties and may cause considerable difficulty in breeding for resistance.

Race 9 again was one of the more important races representing 7.9 percent of all isolates and appearing among isolates from collections made in 15 States. If the related races, 10, 13, 20, and 31, are grouped with race 9 it gains considerably in importance. Race 58 was fifth in abundance and was identified in collections from 16 States. There was evidence, however, that it was in greatest abundance in the Ohio Valley, around the Great Lakes, along the Atlantic Seaboard, and in the Southeastern States.

Besides the geographic distribution noted for race 58 there was evidence that race 93 was most abundant in the Southeastern States, race 44 was most abundant in central United States, and race 11 again appeared in California and Florida. These agree with observations made in other years.

Collections from Kansas yielded the greatest number of races (23) but there were far more collections from that State (97) than from any other State. Collections from Minnesota and Illinois yielded 11 races each, from South Dakota 10 races, and from Alabama and Montana 9 races each. Seven races were isolated only once each and 4 additional races only twice each.

It may be of interest to note that while the variety Lee is ordinarily resistant to races 5 and 16, six cultures of race 5 isolated from collections obtained in 1951 at Choteau and Dutton, Montana, Langdon and Medina, North Dakota, and Rapid City and Underwood, South Dakota, and one culture of race 16 isolated from a specimen collected at Collins, Montana, produced relatively severe infection on seedlings of Lee in the greenhouse at the St. Paul laboratory.

COOPERATIVE INVESTIGATIONS BETWEEN THE DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, UNITED STATES DEPARTMENT OF AGRICULTURE, AND THE KANSAS AND MINNESOTA AGRICULTURAL EXPERIMENT STATIONS

Table 1. Physiologic races of Puccinia rubigo-vera tritici isolated from leaf-rust infected specimens of wheat collected in 27 States of the United States in 1951.

State	Frequency distribution of physiologic races isolated.																													Total No. Isolates	Total No. Races
	1	2	4	5	6	9	10	11	13	15	16	17	18	20	21	28	31	35	44	45	52	56	58	64	77	93	105	114	115	126		
Ala.	-	-	-	42	-	9	-	-	-	5	-	-	-	-	-	2	-	3	-	-	-	-	4	-	-	12	4	-	-	23	104	9
Ark.	-	-	-	1	1	-	-	-	-	1	-	-	-	-	-	5	-	-	-	-	-	-	-	-	-	-	-	-	-	3	11	5
Calif.	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	1
Colo.	-	-	-	1	-	-	-	-	-	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	4	2
Fla.	-	-	-	18	-	1	-	1	-	12	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	3	36	6
Idaho	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	3	-	-	-	-	-	-	-	5	2
Ill.	-	1	-	2	-	3	-	-	-	2	-	-	-	-	-	-	-	1	1	-	-	-	6	1	-	1	3	-	-	7	28	11
Ind.	-	-	-	9	-	5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	2	17	4
Iowa	-	-	-	5	-	3	-	-	-	-	-	-	-	-	-	-	-	2	1	-	-	-	-	-	-	-	-	-	-	5	16	5
Kan.	1	23	-	64	44	32	6	-	6	19	-	1	-	21	1	5	1	1	-	4	47	-	4	1	7	-	17	1	1	31	338	23
Ky.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	1	-	-	-	-	-	-	2	5	4
Mich.	-	-	-	9	-	3	-	-	-	5	-	-	-	-	-	-	-	-	6	-	-	-	9	-	-	-	-	-	-	3	35	6
Minn.	-	1	1	17	1	1	-	-	-	7	1	-	-	-	-	-	-	-	-	-	9	1	2	-	-	-	-	-	-	3	44	11
Miss.	-	-	-	3	-	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	6	3
Mo.	-	-	-	8	-	-	-	-	-	1	-	-	-	-	-	-	-	-	1	-	-	-	1	-	-	1	-	-	-	1	13	6
Mont.	-	3	-	4	-	2	-	-	-	6	1	-	1	-	-	-	-	2	-	-	2	-	-	-	-	-	-	-	-	1	22	9
Nebr.	-	1	-	25	-	3	-	-	-	3	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	2	-	-	4	39	7
N. Y.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	4	-	-	-	2	-	-	3	10	4
N. Dak.	-	2	-	9	-	-	-	-	-	7	-	-	1	-	-	-	-	-	-	-	6	-	1	-	-	-	-	-	-	2	28	7
Ohio	-	-	-	1	-	1	-	-	-	3	-	-	-	-	-	-	-	-	-	-	-	-	17	-	-	-	-	-	-	1	23	5
Okla.	-	-	-	2	-	2	-	-	-	1	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	3	-	-	14	23	6
S. Car.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	-	-	-	-	2	1
S. Dak.	-	3	-	11	-	3	-	-	-	12	1	-	-	-	-	-	-	1	-	-	9	1	-	-	-	-	3	-	-	2	46	10
Tex.	-	-	-	7	-	1	-	-	-	5	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	2	-	-	2	18	6
Va.	-	-	-	4	-	-	-	-	-	3	-	-	-	-	-	-	-	-	-	-	-	-	4	-	-	-	1	-	-	4	16	5
W. Va.	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	1	-	-	-	3	-	-	-	-	-	-	1	6	4
Wis.	-	-	-	2	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	5	8	3
	1	34	1	244	46	71	6	2	6	97	3	1	2	21	1	12	1	11	14	6	74	2	63	2	7	14	37	1	1	123	904	30
Percent of total	0.1	3.8	0.1	27.0	5.1	7.9	0.7	0.2	0.7	10.7	0.3	0.1	0.2	2.3	0.1	1.3	0.1	1.2	1.6	0.7	8.2	0.2	7.0	0.2	0.8	1.5	4.1	0.1	0.1	13.6		

DOWNY MILDEW (SCLEROSPORA MACROSPORA) OF OATS IN THE SOUTH

Thomas E. Summers

Since 1941 when Miles (3) first reported Sclerospora macrospora attacking oats in Mississippi, the downy mildew disease of oats has been reported in Arkansas, Colorado, Georgia, Idaho, Indiana, Louisiana, and Michigan (8, 6, 8, 7, 1, 2, 5). It was found in Alabama and Tennessee during the late spring of 1952. A diseased specimen received from Dr. Robert Earhart indicates that the disease also was present in Florida in 1952. A brief survey conducted in 1940 and reported by Miles and Epps (4) revealed the presence of the disease in the Mississippi counties of Sunflower, Sharkey, Leflore, Washington, and Oktibbeha. There were no published reports of the occurrence of the disease in Mississippi between 1942 and 1951, although it is known to have been present in certain years during that period. Though the presence of the disease in the South in 1951 was known (8), it was not possible to make a comprehensive survey at the time the disease could have been detected most easily. The best time is between the ripening and harvest of normal, healthy oat plants. Diseased plants remain green for several days after the normal plants have ripened and then can be readily observed.

Distribution in 1952

A survey was made in early June of 1952 to determine the distribution of the disease and to ascertain whether economically important losses were caused by the downy mildew disease of oats in the Mid-South. In Mississippi, 85 percent of the oat fields examined contained plants infected by S. macrospora. The severity of infection in different fields ranged from a trace to as many as 25 percent of the plants. In spots in certain fields practically 100 percent of the plants were infected. The disease was severe enough to cause significant losses in grain production in the following Mississippi counties: Claiborne, Warren, Yazoo, Issaquena, Sharkey, Humphreys, Washington, Sunflower, Leflore, Bolivar, Tallahatchie, and Coahoma. All of these counties are in, or partly in, the Delta area of Mississippi where yields were generally good despite losses from the downy mildew disease. Since infected plants produce no seed, the percent of infected plants may be considered as the percent of grain lost in a field. It is estimated that losses due to the disease in this area of Mississippi amounted to more than 200,000 bushels of grain in 1952. The fact that the disease occurs most heavily on fertile soil of high moisture content, where yields are usually good, may partly account for the little attention the disease has received. Another factor contributing to the failure to observe the disease is that the diseased plants in many cases are so distorted that they are often mistaken for weeds or other grasses. Other counties in which the disease was observed but in which no fields examined had more than 2 percent infected plants were : Attala, Winston, Noxubee, Oktibbeha, Lowndes, Webster, Clay, Monroe, Chickasaw, Calhoun, Lee, and Marshall. The disease may have been present in other counties of Mississippi that were not surveyed because of the small acreage of oats grown for grain.

Only a few oat fields were observed in Alabama, but in one, a field near Talladega, more than 60 percent of the plants were diseased and the diseased plants were uniformly distributed throughout the field. Other fields nearby contained from 5 to 10 percent of diseased plants. The disease was observed in Pickens, Talladega, and Calhoun counties in Alabama.

Dr. C. Roy Adair has observed the downy mildew disease of oats in the Stuttgart, Arkansas area for a number of years. More than 15 percent of the plants in a small plot of DeSoto oats grown at the Rice Branch Experiment Station were infected this spring. The writer observed the disease in Chicot, Desha, Drew, Lincoln, Jefferson, Arkansas, Monroe, and Phillips Counties in Arkansas. Severity of infection ranged up to an estimated 7 percent in certain fields south of Pine Bluff, Arkansas, with some reduction in grain yields. This survey was too limited to estimate the losses from the disease for the entire State.

In Louisiana oat plants affected by the downy mildew disease were found in each of four parishes, viz: Madison, Carroll, West Carroll, and Morehouse. The disease was prevalent and severe near Thomastown in Madison Parish, where one 75-acre block of Fulgrain oats contained an estimated 15 percent of infected plants. Other fields in the area were affected to a lesser degree. In the other three parishes no fields with high soil moisture were observed, and damage was slight in all fields examined. Harvest was practically complete in Louisiana at the time the survey was made.

A few plants of an undetermined variety of oats were found to be infected in Hardeman County, Tennessee, about 10 miles north of the Mississippi State line. No other areas of Tennessee were visited.

Diseased plants were observed in late May in Carroll County, Georgia, but no systematic survey was conducted in that State.

Known Susceptible Varieties

Prior to this report Brunker, Colburt, Fulghum, Kanota, and Victoria (6); Overland (7); Taggart and Delair (8); C.I. 4316 (2); C.I. 3664 and C.I. 3909 (D69 x Bond) (5) had been reported to have been attacked by S. macrospora in the United States. Other commercial varieties that were attacked this spring include Delair, Arlington, Delta Red 88, DeSoto, Fulgrain, Mustang, Nortex 107, Stanton, Taggart, and Victorgrain.

Field Distribution of Diseased Plants

All of the symptoms of the disease described by Caldwell et al. (1), and Miles and Epps (4) were observed during this survey. The distribution of diseased plants within a field varied considerably. In some fields the plants were either sparsely or rather uniformly infected while others were uniformly lightly infected except in low areas where nearly all plants were diseased. In a few fields infection apparently was limited to a narrow strip along the edge of the field adjacent to areas in which wild grasses susceptible to the disease were growing.

Sources of Infection and Control

Preliminary greenhouse and field plot experiments, and observations of the behavior of the disease in the field, indicate that two sources of initial inoculum are involved. Seed oats from fields containing diseased plants may have been contaminated by oospores or by bits of diseased plant tissue containing oospores during threshing. Fields seeded with such oats become uniformly infected lightly or, parts of the field favorable to the development of the disease may show heavy infection. The second source of primary inoculum is from the fungus persisting in infested soil. The oospores may persist in debris and diseased plant tissue of any of the small grains or in certain wild grasses known to be susceptible to S. macrospora. Where a wild grass host is involved infection usually is limited to a relatively narrow strip adjacent to ditch banks, field roads, or similar areas where the wild host grows abundantly.

Until varieties of oats resistant to S. macrospora are found or developed it is suggested that seed oats for sowing in the delta lands of Arkansas, Louisiana, and Mississippi be procured from areas or fields known to be free of the disease. The grain from heavily infected areas of the fields should not be used for seed in harvesting fields certified for seed production.

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DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, STATE COLLEGE, MISSISSIPPI

SEPTORIA BLACK STEM OF OATS ABUNDANT IN MOST OF THE NORTH CENTRAL STATES

T. R. Stanton[1]

Septoria black stem (Leptosphaeria avenaria Weber) (Septoria leaf blotch or speckled blotch) in epidemic proportions was noted by the writer in many of the important oat-growing sections of Iowa, the Dakotas, Minnesota, and Wisconsin during an automobile trip from June 29 to July 16 with representatives of the Quaker Oats Company. Septoria black stem was observed in Illinois also, but in the cash grain sections visited the disease apparently developed too late to do much damage to oats. This disease undoubtedly will reduce the yield and quality of oats in many sections in 1952. Some Septoria black stem was reported in 1950 and still more in 1951. Since it has now attained epidemic proportions over wide areas it must be placed in the category of a major oat disease.

In certain yield-test nurseries Septoria black stem was so severe on some varieties and selections as to cause 100 percent lodging. These plots had the appearance of oats that had stood in the field after becoming fully ripe during a long period of wet weather. The leaves were spotted or completely dried up and the stems or culms had turned dark brown or almost black and were broken over. Many of the lemmas also were badly infected by the disease and to make the situation more serious some of the caryopses likewise were infected and had turned black.

It now appears that much of the commercial oat supply from several of the big-producing States has been rendered unfit for milling because of the presence of these black groats. Just a few discolored groats in a commercial lot of grain renders them unsuitable for processing into rolled oats for breakfast food. Since oats free from black groats are likely to be mixed with damaged lots carrying disease-colored groats in elevators, cars, and trucks it may be very difficult to purchase oats at the terminal markets that are entirely free from black groats.

Samples collected from farmers and elevators in Iowa and Minnesota, where most of the marketed oats are produced, nearly all contained at least a few black groats. This shows the wide prevalence of Septoria black stem.

There was much less Septoria black stem in the eastern half of South Dakota although Cherokee, which is rather susceptible, was the dominant variety grown there. The most destructive epidemic observed was in a yield-test nursery at Hancock, Wisconsin, where nearly all varieties and selections were heavily infected with Septoria black stem. There were observable differences between varieties and selections. Some of the older sorts such as State Pride, and Spooner appeared to be injured less at that time than were most of the newer varieties, which included many Bond derivatives.

Dr. H. C. Murphy has described the development of this disease as follows: "Septoria black stem has two distinct phases in the field. It is first evident in causing dark, necrotic leaf spotting of the lower leaves of plants early in the season. The black stem phase appears sometime after heading and becomes more severe as the plants near maturity. This secondary phase is characterized by the blackening of the upper plant internodes. As a result of these culm lesions certain varieties and selections have shown extreme lodging at the points of infection." The black stem stage developed to an extreme degree in many sections in 1952, apparently where there was an early onset of the disease. In the absence of germplasm varieties carrying satisfactory resistance to Septoria black stem are not yet known so that the outlook for its control through breeding is not promising. Furthermore, not much is known about the disease organism. Additional research is needed badly.

DIVISION OF CEREAL CROPS AND DISEASES

[1]Collaborator in Oat Investigations, Division of Cereal Crops and Diseases.

GENERAL CORN RUST AND LEAF BLIGHT INFECTION IN WISCONSIN

James G. Dickson

A general spore shower of Puccinia sorghi and Helminthosporium turcicum was indicated by the extensive appearance of the two diseases in southern Wisconsin about July 25. Based on the incubation period for these two diseases under controlled environmental conditions, the spore shower occurred approximately July 10 to 12. Both rust and blight were found only on the two leaves that were exposed in the leaf whorl at the indicated time of the spore shower. Spread of the two diseases on very susceptible inbreds and hybrids has occurred since. General spread on commercial hybrids in the area was not evident August 7.

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DIPLODINA ON PEACH IN LOUISIANA

Norman L. Horn

Numerous isolations were made from diseased peach fruit and stems obtained from the North Louisiana Hill Farm Experiment Station, Homer, Louisiana. Also, similar diseased plant material was placed in moisture chambers and incubated at room temperatures. In both instances the same fungus was found consistently associated with the diseased material. The organism was tentatively identified as Diplodina. The disease was found in other orchards near Ruston, Louisiana.

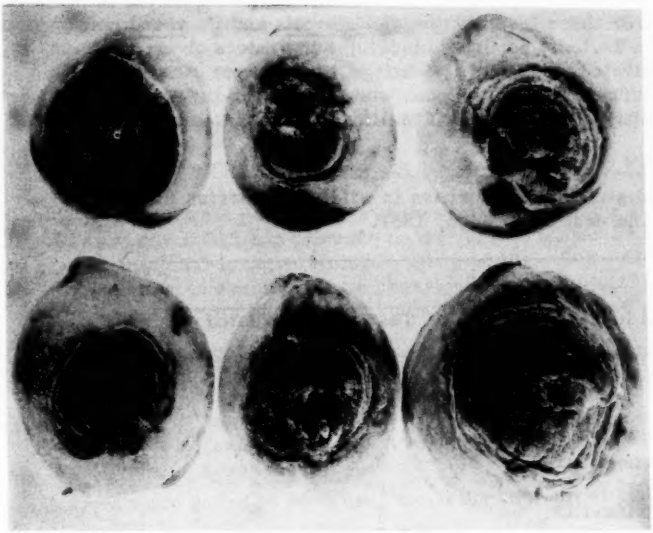

FIGURE 1. Peach fruit infected with Diplodina sp.

The symptoms on the fruit (Fig. 1) when first formed, appeared similar to those caused by the brown rot fungus; that is, under moist conditions the earliest indications of the disease were small, circular, brown spots. These rapidly increased in diameter until finally the whole fruit became involved. The spots did not become sunken but were soft and watery. Shriveling, which occurred in concentric rings, was not evident until the spots were much enlarged. In the folds of the host epidermis, numerous brown, stromatic masses appeared which soon turned black and which became covered with light grey mycelium. This mycelial matting was sometimes of such a texture that its appearance was similar to the spore masses of the Monilia type. However, within the black stromatic masses which protruded through the host epidermis were numerous pycnidia which separated singly when the material was crushed. Later masses of conidia, grey in color, exuded from the fruiting structures. Finally, the fruit became dry and shriveled or mummy-like in form.

The fungus has the following characteristics: fruiting body a pycnidium, glabrous, separate, yet imbedded in a black stroma (they separate upon pressure), not rostrate, not maculicole, and without a clypeus, average size approximately 180 μ in diameter; conidia 2-celled, hyaline, constricted at the septum, average size approximately 28 μ x 6 μ, not catenate and not appendaged; basidia simple. This description follows in part the key of Clements and Shear.

The fungus grew rapidly on potato-dextrose agar and fruited abundantly within seven days.

Fruit was inoculated with the fungus by dipping the peaches in a conidial suspension, one series being punctured with a needle several times before inoculation. Uninoculated fruit was used as controls. After treatment all the fruit was placed in a moisture chamber. Within 24 hours spots appeared on the treated peaches. Approximately five days later the inoculated fruit was almost completely rotted. The injured fruit contained spots not only at the wounded areas but at other locations as well. The disease, therefore, was reproduced and the organism reisolated.

Because this fungus caused a "brown rot" of peaches and because the symptoms appeared similar in some respects to the Sclerotinia [Monilinia] brown rot, it is possible that this fungus has been present in this area for some time and is likely that the disease has been assumed to be the Sclerotinia brown rot. Nevertheless, the fungus is capable of causing a fruit disease of peaches and may be potentially, if not already important in the North Louisiana peach growing area.

DEPARTMENT OF PLANT PATHOLOGY, LOUISIANA AGRICULTURAL EXPERIMENT STATION, BATON ROUGE, LOUISIANA

PRELIMINARY TESTS OF WILD STRAWBERRIES FROM EASTERN OREGON FOR THE PRESENCE OF VIRUS INFECTION[1]

P. W. Miller

In an earlier paper[1], the writer presented data indicating that wild strawberries belonging to the species Fragaria ovalis and F. chiloensis may be important natural sources of virus infection in western Oregon. In these earlier investigations, no wild strawberries from east of the Cascade mountains were tested.

As substantial acreage of strawberry plants is now being grown for certification in eastern Oregon it was thought advisable to determine to what extent various species growing in the wild in eastern Oregon carry viruses. For these investigations, F. ovalis and F. vesca plants from different clones were collected from 6 widely separated locations in eastern Oregon. None of the collections were made near cultivated strawberry plantings, the nearest strawberry field being over 100 miles away. To ascertain whether viruses were present in the plants, they were stolon-grafted to healthy virus-sensitive F. vesca (East Malling strain) indicator plants. The results are given in Table 1.

Table 1. Extent and distribution of viruses in wild strawberries in some areas in eastern Oregon, 1951.

Species and location	Plants	
	Tested	Infected
	Number	Number
F. vesca		
12 miles W. of Seneca	6	0
Near Ochoco	1	0
25 miles N. E. Prineville	9	0
9 miles S. of Canyon City	7	0
20 miles N. of Burns	2	0
F. ovalis		
10 miles W. of Sisters	8	1
25 miles N. E. Prineville	6	0
9 miles S. of Canyon City	2	0
12 miles W. of Seneca	6	0
20 miles N. of Burns	5	0
Near Ochoco	1	0
Total	53	1

The limited data given in Table 1 indicate that virus infection in the native strawberry species in eastern Oregon may be less than west of the Cascades. If further investigations should confirm the results of these preliminary studies, the expansion of the certified and foundation stock strawberry plant industries east of the Cascade Mountains in Oregon might be indicated.

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[1]Miller, P. W., Wild strawberries as a source of strawberry virus infection. Plant Disease Reporter 35: 129-130, 1951.

SURVEY FOR VIRUS-INFECTED WILD STRAWBERRY PLANTS IN EASTERN UNITED STATES

C. P. Marcus, Jr.

Virus-infected wild strawberry plants constitute a possible source of infection of healthy stocks of commercial strawberry varieties. Knowledge as to the amount and distribution of infection in wild plants, therefore, is of considerable value. Such information is important when one is considering the necessity of isolating plantings of cultivated strawberries from wild strawberry plants or their destruction. A survey was therefore begun in the fall of 1951.

P. W. Miller made similar surveys in Oregon and Washington and found virus-infected plants of both the beach strawberry, Fragaria chiloensis, and the western meadow strawberry. F. ovalis, in some cases far from cultivated fields. He suggested that virus may be indigenous in the wild there. In limited tests Miller found only one virus-infected collection in eastern Oregon and suggested that there is less danger of virus infection of the plants there than in the western part of the State.

Source and Nature of Wild Plants

Wild plants from several strawberry-plant-producing areas in the eastern half of the United States were obtained from workers in the various States or collected by the author. A wide range of genetic material is represented in these plants. Some are probably pure Fragaria virginiana, others are hybrids resulting from cross-pollination of wild plants with commercial varieties, others are seedlings of varieties, and still others are plants from abandoned commercial plantings.

The numbers of different lots of plants from the various States were as follows:

Maryland	36	Michigan	6
Delaware	15	Illinois	19
Tennessee.....	14	West Virginia	3
Arkansas	3	Total	96

Collectors were asked to select not more than one lot of plants from each bed of wild plants; therefore each lot of plants represents a different clone.

Procedure in Testing for Presence of Virus

The plants received, mostly in the fall and winter of 1951-52, were potted and labeled according to State and lot number and placed on benches in the greenhouse. They were grown under lights so that runners would be produced quickly. When the runners were of proper size, the plants were indexed by grafting to runners of Fragaria vesca by the method described by Demaree and Marcus.[1]

Results

The results of the indexing given in Table 1 show that 21 out of 94, or 22 percent, of the lots of wild plants were virus-infected and 73 were virus-free. Two lots of plants were not tested.

Discussion and Conclusion

It is evident from this survey that virus-infected wild strawberry plants constitute a source of infection for virus-free commercial strawberry plantings. Therefore, it is obvious that precautions such as isolation of commercial plantings or elimination of wild plants should be undertaken. The type of precaution to be used will vary with different areas of the country. For example, on the Delmarva peninsula, where strawberries have been grown extensively for many years, it is difficult to find a planting site which is not near wild plants or other strawberry plantings. In this area it would be advisable to eliminate wild plants by the use of some type of weed killer or by removal. It has been found very easy to eradicate wild strawberries from the vicinity of plant-propagating fields either by digging or by the use of chemicals. In an area such

[1] Demaree, J. B., and C. P. Marcus, Jr. 1951. Virus diseases of strawberries in the United States, with special reference to distribution, indexing, and insect vectors in the East. Plant Dis. Reptr. 35: 527-537.

Table 1. Results of indexing wild strawberry plants.

State	No virus	Virus 1[a]	Virus 2[a]	Virus 1 and 2[a]
	Number	Number	Number	Number
Maryland	27	3	2	3
Delaware	9	5	1	0
Arkansas	2	0	1	0
Michigan	5	0	1	0
Illinois	16	2	0	0
West Virginia	3	0	0	0
Tennessee	11	0	3	0

[a]Virus types described in paper listed in footnote number one.

as Tennessee, where there is much virgin land available for strawberry plantings, it should be less difficult to isolate the plantings from other commercial plantings or wild plants. At the present time it is recommended that virus-free plantings be isolated by at least 3,000 feet from wild or other commercial strawberry fields.

The virus-free plants may have escaped infection or there is a bare possibility that some of them may be immune or klendusic (possess a quality that hinders inoculation). The clean plants are being tested for immunity and klendusity.

A test of maintaining virus-free stocks is now being made in the West Virginia area, where no virus-infected plants were found in the very limited tests.

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PHYSALOSPORA ILICIS ON ROTUNDIFOLIA HOLLY IN GEORGIA

Charles H. Driver

Leaves of rotundifolia holly (Ilex crenata var. rotundifolia Hort.) heavily infected with Phyllosticta ilicicola (Cke. & Ell.) Ell. & Ev., the conidial stage of Physalospora ilicis (Schleich. ex Fr.) Sacc., were collected during the early part of September 1951, from dying plants on the Emory University campus, near Atlanta (Fig.1). In the area in which the collection was made 17 out of 20 plants died within 30 days after the disease was first observed. The heavily infected plants were three years old and had been growing for one year in a landscape planting near a newly constructed building. All plants had been purchased from a local nursery northwest of Atlanta. The nurseryman had propagated and grown the plants himself. Other plants of the same variety set nearby some five years earlier exhibited only very light leaf spotting symptoms.

FIGURE 1. Rotundifolia holly leaves with pycnidia of Physalospora ilicis.

When first observed, a few leaves had dropped from the heavily infected plants. Later the leaves remaining rapidly lost their characteristic deep green color, turned dark grey and examination revealed many pycnidia visible through the upper epidermis. The disease symptoms progressed from the top down and eventually all of the leaves were shed, leaving the dead defoliated stems (Fig. 2).

The numerous pycnidia which occurred on both surfaces of the leaves were black, measuring 164-172 x 148-164 (166x 155) μ, and at first were embedded in the leaf tissues with a small papillate ostiole extending to the surface. Conidia were numerous, ellipsoid to oval to pyriform, hyaline, granular within, variable in size, measuring 8.5-11.5 x 5-7 (5 x 10) μ. Perithecia of the perfect stage were found in May 1952 on a few leaves that had overwintered on the ground around the dead plants. The fungus was isolated from infected leaf sections on carrot agar and both the perfect and imperfect spore stages were produced in culture on corn meal agar.

FIGURE 2. Rotundifolia holly plant exhibiting symptoms of heavy infection by Physalospora ilicis.

Apparently this is the first time that P. ilicis has been reported on this variety of holly. Weiss[1], however, recorded it as having been reported on Ilex crenata Thumb.

Specimens of this disease have been filed in the following herbaria: C. H. D. #869, Department of Plant Pathology, University of Georgia; C. H. D. #873, Department of Plant Pathology, Cornell University; and C. H. D. #873, Division of Mycology and Disease Survey, U. S. Department of Agriculture, Beltsville, Maryland.

The results of a cytotaxonomic study of this fungus will be published at a later date.

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[1]Weiss, Freeman, 1950. Index of plant diseases of the United States. Plant Disease Survey Special Publication, Number 1, Part 1, 218 pp., (p. 50).

VIRUS YELLOWS OF BEET IN THE UNITED STATES

G. H. Coons

Virus yellows has been known in Europe for many years as a serious disease of sugar and forage beets. In 1950 and 1951, two European specialists[1] who examined yellowed sugar beet plants from Michigan, Colorado, Utah, Oregon, and California, stated that the disease symptoms were similar to those shown by European sugar beets affected by virus yellows.

Positive statement can now be made that virus yellows of beets is widespread in the United States. The evidence to be presented consists of (1) results from serological tests in which rabbit antiserum of virus yellows gave positive reactions against juices from leaves of suspected sugar beet plants collected in the States mentioned above; (2) positive results in the mechanical transmission of virus yellows in which juices from leaves of affected plants collected in Michigan and California were used as the sources of the virus; (3) positive results in aphid transmission tests, in which typical virus yellows symptoms appeared in seedling sugar beets inoculated with aphids (Myzus persicae Sulz.) that had fed on yellowed plants collected in California and Oregon; (4) confirmation of virus yellows diagnoses in (2) and (3) by positive reactions with virus yellows antiserum in tests of juices from the leaves of the inoculated plants in which virus yellows symptoms developed; and (5) serial transmission of virus yellows to sugar beet seedlings by means of aphids, the virus sources being the affected test plants from earlier aphid transmission tests.

The serological tests of suspected sugar beets were made with antiserum of virus yellows contributed by the Instituut voor Rationele Suikerproductie, Bergen-op-Zoom, Netherlands, and brought to us by its director, Dr. Henk Rietberg. The serum was prepared by the Laboratorium voor Bloembollenonderzoek, Lisse, Netherlands. Acknowledgment is made to its director, Dr. E. Van Slogteren, for supplying the high-titre antiserum obtained at Lisse by sensitizing rabbits against the common strain of virus yellows of the Netherlands, as well as another high-titre serum reactive against a strain of virus yellows which tends strongly to clear the veinlets of the affected sugar beet plants. In all, three separate shipments of antisera were made available for the tests in the United States, representing three series of rabbit immunizations with juices from leaves of affected sugar beet plants collected in the Netherlands and comprising both virus strains. In addition, antiserum of healthy sugar beets -- that is, serum from rabbits immunized with juices from leaves of known healthy sugar beet plants -- and normal rabbit serum were furnished for the tests. Methods of producing the antisera of titre high enough for use in testing for presence of sugar beet virus yellows in juice of suspected plants and the techniques of testing were developed at the Lisse laboratory (1).

The Lisse technique for testing suspected sugar beet plants for virus yellows as used at Bergen-op-Zoom institute was outlined by Dr. Rietberg and is as follows:

> (1) Absorption of antigens of normal sugar beet leaves and their removal are accomplished by mixing the antiserum of virus yellows at a 1:10 ratio with juice pressed from healthy sugar beet leaves, incubation for 2 hours at 37°C., and storage overnight at about 0°C. The mixture is then centrifuged at 10,000 to 13,000 r.p.m. until clear. (2) Juice from leaves of a suspected plant is pressed out, diluted 1:1 with 0.9% NaCl solution, and centrifuged at fairly high speed until clear. (3) Minute drops of clear, diluted antiserum and of the juice to be tested are placed side by side on a cover glass, mixed with a small glass rod, and then inverted over a Van Tieghem cell. The cover glass is sealed to the ring with vaseline. (4) After 1- and 2-hour periods of 37°C., respectively, the drops are examined under low power of a darkfield microscope. A positive reaction manifests itself by formation of clouds or flocculae -- usually definite at the first reading, sharp at the second. (5) Each test of diluted antiserum vs. diluted antigen is checked by similar preparations in which antiserum of healthy beets and normal rabbit serum replace the antiserum of virus yellows. Such preparations must be free of clouds or flocculae for a positive test with the yellows antiserum to be accepted. (6) As a check on conditions of the test, juice from a sugar beet plant known to be affected with virus yellows is included in each set of tests. It must react positively to the virus yellows antiserum. This juice must also give negative readings against anti-healthy and normal sera.

The Station Central de Pathologie Végétale, Versailles, France, through its director, Dr.

[1] Dr. Henk Rietberg, Director, Instituut voor Rationele Suikerproductie, Bergen-op-Zoom, Netherlands; and Dr. Raymond Hull, Rothamsted Experimental Station, Dunholme, England.

Pierre Limasset, kindly supplied vials of its antiserum of virus yellows as produced by immunizing rabbits with juices from sugar beets affected with virus yellows as it occurs in France. In January 1951, Dr. Limasset demonstrated his method with juices from sugar beet plants collected in the United States.

The method used by Limasset (2) is essentially similar to that used at Bergen-op-Zoom, except that larger drops of dilute antiserum and antigen are placed at the center of a microscope slide, mixed, and then covered with a cover glass. As a control, a drop of either dilute normal rabbit serum or antiserum of healthy sugar beets either absorbed as described above or diluted 1:15, is placed at the side on the same slide and covered with a cover glass. The slides are held in moist chambers at 20°C. for 20 and 40 minutes for the first and second readings. The preparations are examined with the darkfield microscope. Presence of clouds and flocculae with antiserum of virus yellows indicates a positive reaction, but the control preparation must be clear.

Sugar beet plants collected near Cone, Michigan, in 1950 -- suspected of being affected with virus yellows -- were the first to give positive serological reactions when tested in February 1951 under Dr. Rietberg's direction (3). During the next two months, 26 sugar beet plants collected near Fort Collins and Longmont, Colorado, which had been noted as either showing symptoms or as symptomless, or as being affected with mosaic or savoy, were tested with rabbit antiserum of virus yellows. Nine of the plants gave positive reactions, two were probably positive, and fifteen were negative. In general, the field diagnoses were substantiated by the serological tests. Plants showing both virus yellows and mosaic symptoms gave positive tests for virus yellows. Sugar beets affected with mosaic but showing no signs of virus yellows were negative in reaction. Colorado as well as Michigan plants affected with savoy but showing no symptoms of virus yellows gave negative reactions.

Two monogerm sugar beets that had been received from Salt Lake City in 1950 had been maintained in the greenhouse at Plant Industry Station, Beltsville, Maryland, by cloning. These clones gave positive reactions. There was no evidence that the disease had spread to neighboring plants. Dr. Hull had noted what appeared to be virus yellows as present in the sugar plant greenhouse at Salt Lake City, Utah, in August 1951. A yellowing typical of virus yellows was found in sugar beets adjacent to the greenhouse, but not in commercial sugar beets at a distance from the greenhouse. Such diseased plants, when brought to Beltsville, have given positive tests with yellows antiserum. Examination of the plants at Salt Lake City by Dr. Rietberg in February 1952 confirmed Dr. Hull's diagnosis. Leaves of sugar beet sent from the Salt Lake City greenhouse by Dr. F. V. Owen on January 21, 1952, gave strongly positive reactions against rabbit antiserum of virus yellows.

Sugar and red garden beet plants were collected in Oregon in August 1951 by Dr. Hull as virus yellows suspects. In California, the yellowing of sugar beet fields in vicinity of Salinas had been noted for a number of years. The condition was called locally "Salinas yellows", and was ascribed to soil conditions or other causes. Virus yellows had not been considered the cause. Dr. Hull definitely stated the symptoms were those of virus yellows as he knew it in England. Dr. J. S. McFarlane sent sugar beets from three fields near Salinas, California, which Dr. Hull stated showed typical symptoms: Jensen, Jacobs, and Salinas Valley Vegetable Growers Exchange fields. The last-named was late planted and the time of Dr. Hull's visit showed yellowed areas surrounded by apparently green, unaffected plants. When the field was observed on October 22, 1951, it was 100 percent yellowed. Leaves taken from the Oregon and California plants have uniformly given positive reactions with the virus yellows antisera.

Since the first tests, in which experience in the technique was being gained, many hundred tests have been performed involving at least 100 different sugar and red garden beet plants from Michigan, Colorado, Utah, Oregon, Washington, and California. Many of the individual plants have been tested repeatedly and with excellent concordance among the tests.

In this extensive series of tests, positively reacting plants have been found in all collections. The conformance of positive serological reaction with definite diagnostic symptoms -- i.e. brittle, thickened, yellow outer leaves -- has been entirely satisfactory. Some tests have been lost from spontaneous flocculations in the drops. Such occurrences are usually traceable to laboratory temperatures higher than 55°F. and to some drying of the minute drops during their preparation. Sugar beet plants that have grown under very warm conditions in the greenhouse are usually unsuitable for the serological tests.

In the serological tests, in addition to the two stocks of antisera originally supplied, the Lisse laboratory -- again through the good offices of Dr. Rietberg -- supplied a third stock of very high-titre antisera prepared September 3, 1951. The reactions obtained with the new antisera were very strong. The antiserum prepared with the common strain of virus yellows of Netherlands was equally reactive with virus yellows in plants collected in the United States as the one prepared with

the vein-clearing strain. The two antisera could be used interchangeably. Here again, the new rabbit antisera were non-reactive against beet mosaic, savoy, and yellowing that had come from causes other than virus yellows. No reaction was obtained with juice from leaves from sugar beets affected with curly top.

Rabbit antisera of virus yellows prepared in Versailles, France, were tested in January 1952 by Dr. Limasset, who kindly brought to the United States several lots of antisera. The tests were run with juices from leaves of yellowed California sugar beets, as sent in by McFarlane. Parallel preparations were made with virus yellows antiserum from Lisse, Netherlands. The juice from leaves of the "Salinas yellows" plants reacted positively with antisera of virus yellows from both France and Netherlands, the companion tests being in satisfactory concordance. The controls, of course, remained clear.

All European scientists who have used the serological method for diagnosis of virus yellows of sugar beet and its close botanical allies are unanimous in stating that the reaction is highly specific and that leaves yellowed by other factors such as mosaic, nutritional or moisture conditions, do not give positive reactions. A positive reaction may be accepted as definite proof of the presence of the virus, whereas a negative test does not exclude the presence of the yellows virus since the test requires that a considerable quantity be present in order to be detectable (4).

As part of all tests, juices from healthy plants -- usually plants grown in the field or greenhouse at Beltsville from seed -- have been tested against the various antisera. No positive reactions have been obtained. As stated, other causes of yellowing of sugar beet leaves have uniformly been found to be non-reactive with the antiserum of virus yellows. The yellow, flaccid lower leaves that become very common in winter on greenhouse-grown sugar beets have been repeatedly tested with negative results. On the other hand, juice from older leaves of inoculated or naturally infected sugar beet plants that show the yellow and brittle leaves characteristic of virus yellows, have given positive serological reactions with antisera produced by immunizing rabbits with juice from leaves of sugar beets affected with strains of virus yellows that occur in Netherlands and France. The writer has no reluctance in accepting the judgment of European investigators as to the specificity of the reaction to virus yellows antiserum or the diagnostic significance of positive reactions with such antisera.

Kassanis (5) reported that mechanical inoculations with virus yellows resulted in about 10 percent of the plants becoming systemically infected. When plants were kept four days in the dark, three times as many became so infected. Juice inoculations were made with sap from leaves from a Cone, Michigan, sugar beet that had given positive serological tests with antiserum of virus yellows. The juice was rubbed on leaves of healthy sugar beets following a light dusting of the leaf surfaces with carborundum powder. In the first experiment, one plant in fourteen inoculated showed a yellow spot one week after inoculation, and similar yellowed spots and blotches appeared on the new leaves that developed on the plant. The check plants remained healthy. Juice pressed from a blotched leaf when tested serologically gave a positive test. In a second experiment, sugar beet seedlings were inoculated with juice from the leaves of the same Michigan plant. The seedlings had been kept in the dark in a refrigerated cold-storage room for a week. The leaf surfaces were dusted with carborundum powder. Nine plants were inoculated by a light rubbing with juice from the Michigan plant; three were rubbed with water to serve as checks, ten untreated plants of the same variety that stood nearby constituting additional check plants. After about three weeks, all plants except one were normal. This plant was noted as having suspicious mottling not unlike mosaic, but occurring everywhere throughout the leaves regardless of age. Its leaves had a very firm texture. Juice was squeezed from the leaves of this plant on June 8, 1951, frozen, and taken in frozen condition to the Bergen-op-Zoom laboratory. Positive readings with antiserum of virus yellows were obtained at the Bergen-op-Zoom laboratory by Dr. J. A. Hijner, whereas juice from the leaves of a companion check plant was negative in reaction.

Other attempts to transmit virus yellows mechanically have been made. In two experiments, no positive symptoms have been detected and the experiments have been noted as failing to show transmission. It was noted that these plants were rather old, had tough leaves, and were growing slowly. In two other experiments, a limited number of transmissions have occurred. The usual technique was employed, namely the leaf surfaces were dusted lightly with carborundum powder and then gently rubbed with a cotton swab saturated with juice plus macerated tissue from yellow leaves of plants that had been found to be serologically positive for virus yellows. As checks, plants were treated similarly except the swab was saturated with material from leaves of healthy plants.

In one juice transmission experiment, leaves from sugar beets suspected of being affected with virus yellows were collected by Dr. Hull in the vicinity of Bay City, Michigan, September 14, and brought to Beltsville, Maryland. These had been held in the refrigerator until September

29. Five groups of leaves of the collection had reacted positively to antiserum of virus yellows. Juices from three of these (B1, C2, and C6) were used to inoculate healthy sugar beet seedlings. The results were as follows:

Inoculated		Diseased with virus yellows
B1	23 plants	1 plant
C2	22 plants	2 plants
C6	23 plants	7 plants
Check	23 plants (rubbed with juice from healthy beet)	0 plants

On April 18, 1952, another experiment in mechanical transmission of virus yellows was performed, using the same technique. Juice was taken from a sugar beet plant showing definite virus yellows symptoms as a result of inoculation by aphids that had been colonized on a "Salinas yellows" plant from the Jensen field, Salinas, California. The juice from the leaves of this plant had shown positive serological reaction with virus yellows antiserum.

Sugar beet seedlings in the 2- to 4-leaf stage and in the 4- to 6-leaf stage were used as test plants in this experiment. Fifteen plants of each age were rubbed on both dorsal and ventral leaf surfaces with juice from healthy leaves to serve as checks. Under two months of observation, these check plants remained healthy. Of the fifteen young plants inoculated mechanically with juice from the inoculated test plant with virus yellows symptoms, eleven showed positive symptoms of virus yellows, the older leaves becoming yellow and brittle. Of the fifteen older plants similarly inoculated, three became definitely diseased with virus yellows. Three representative plants were checked serologically and gave positive reactions against antiserum of virus yellows.

The results from mechanical inoculations are, in line with Kassanis' report, variable. It is not clear that age of plants is a decisive factor, although the differences in the April 18 experiment are suggestive. From the tests that failed completely to show transmission, it was clear that plants with old, tough leaves are not favorable subjects for transmission tests.

Transmission of virus yellows by the green peach aphid (Myzus persicae Sulz.) has been accomplished with both Oregon and California sugar beet plants as virus sources. In his visit to the western sugar beet areas in August 1951, Dr. Hull found both red garden and sugar beets in Oregon that he suspected as having virus yellows. As previously stated, in California Dr. Hull found most fields entirely yellow with apparently 100 percent infection, but a late-planted field (Salinas Valley Vegetable Growers Exchange field) had yellowed areas surrounded by green, apparently normal plants. Dr. J. S. McFarlane sent sugar beet roots from three fields to Beltsville, Maryland. Roots selected at random were used as inoculation sources in the tests to be reported.

The Oregon sugar beet designated by Hull as probably affected with virus yellows had virus-free green peach aphids[2] colonized on it for about 3 weeks. On November 14, 1951, outer leaves heavily infested with the insect were cut into small pieces, each carrying about ten apterous aphids. By means of a small clip-on cage fastened so that the undersurface of the leaf was exposed, nine healthy sugar beet seedlings (4-leaf stage) were inoculated, using about ten aphids per plant. The aphids promptly began to feed on the sugar beet leaves. Three days later the cages were removed and the plants were sprayed with nicotine to kill the aphids.

On December 12, four of the nine inoculated plants showed typical symptoms of virus yellows as it appears in winter in the greenhouse, namely yellowed older leaves that are slightly thickened and of brittle texture. Re-check on January 29, 1952, showed one additional plant with typical symptoms. Three of the plants diagnosed as having virus yellows were tested with antiserum of virus yellows and gave positive reactions. The seven check plants in this experiment not exposed to aphids remained healthy.

On December 13, 1951, non-viruliferous green peach aphids that had been colonized on a "Salinas yellows" sugar beet from the Jensen field near Salinas, California, were transferred to healthy sugar beet seedlings in the 2-leaf stage. Older yellowed leaves heavily infested with aphids were cut into small pieces, so that each piece carried about ten aphids. Each healthy sugar beet seedling was inoculated by placing a piece of the beet leaf with the aphids upon it, the plant then being covered with a glass chimney. Yellowed lower leaves were taken from a sugar beet collected in the Jacobs field near Salinas, California, also noted as showing "Salinas yellows" Non-viruliferous green peach aphids from the stock culture on a known healthy sugar beet were placed on

[2]Green peach aphids (Myzus persicae Sulz.) were supplied by Dr. Floyd F. Smith, Bureau of Entomology and Plant Quarantine, from his stock cultures.

the detached leaves. After a few hours' feeding, these aphids were allowed to move to the healthy sugar beet seedlings, ten aphids per plant. Glass chimneys were placed over these plants to confine the aphids. Next day the aphids on the seedling sugar beets were noted as feeding, and the aphids were then killed by nicotine spray.

Results were recorded on January 29 and January 31. Such plants as were considered doubtful because symptoms were not clear-cut because outer leaves had died, or the whole plant was wilted, were re-examined in two weeks and final decisions made. The results as given in Table 1 represent the decision as to virus yellows based on plant symptoms as checked by the later reading. On January 31 and February 1, all plants were tested serologically. The results of these tests are also given in Table 1. This table shows that of ten test plants inoculated by means of aphids with inoculum from a sugar beet from the Jensen field, seven were diagnosed as affected with virus yellows. The serological tests agree completely with respect to the three plants not showing virus yellows symptoms. These plants were negative in the serum tests. One plant was marked doubtful in the serum test, but positive as to symptoms. This could merely mean that the quantity of virus in the leaves chosen was not adequate for the serum test. It does not contradict the finding based upon symptoms.

Of the nine plants from the Jacobs field, six plants were noted as affected with virus yellows. Here again the serum tests conform to the diagnoses, plants that are negative in symptoms being negative in the serum tests. One plant diagnosed as positive gave a negative serum test, and another a doubtful reading. Such readings are taken to indicate lack of enough virus in the sample tested to give a reaction, rather than a contradiction of the diagnosis.

The eleven check plants did not show virus yellows symptoms, and in no case was a positive reaction with antiserum obtained.

On January 8, another transmission test of "Salinas yellows" was conducted using a sugar beet from the Salinas Vegetable Growers Exchange field. Dr. J. S. McFarlane took roots from the field in early September shortly after Dr. Hull's visit, and one of these roots taken at random was caged and non-viruliferous green peach aphids placed on the developing leaves. After about a month, this plant became heavily infested with aphids.

Healthy sugar beet seedlings in the 2- to 4-leaf stage were used as test plants. Each plant was covered with a lamp chimney cage. Inoculation was made by placing leaves or portions of leaves from the "Salinas yellows" sugar beet on the seedling sugar beets, so that the aphids could crawl from the old leaves to the young plants. About 25 aphids per plant were used. The check plants in this test were never inoculated with aphids but remained in the greenhouse during the infection period of the inoculated plants. The aphids were noted as feeding on January 9, and then were killed by nicotine spray.

No symptoms of virus yellows were noted on January 29, and final reading was made March 21, decision of presence of virus yellows being based on brittle yellow leaves of the type characteristic of virus yellows under greenhouse conditions. Serological tests were made on January 29 (3 plants), March 21, April 1, 2 and 3. The results are given in Table 2.

Of the 21 plants inoculated by the green peach aphid, 13 were diagnosed as positive as to virus yellows symptoms. Twelve of these in an independent reading with antiserum of virus yellows gave a positive reading, and one was lost. In 7 cases, the reading was particularly strong. Of the 21 inoculated, 7 plants did not show virus yellows symptoms, and 5 of these were negative to the antiserum. Two were weakly positive or doubtful. It would take further study of these plants to determine if virus yellows was present or not. One plant was marked as doubtful as to symptoms. It did not give a clear-cut reading with the antisera, being negative to the Dutch antiserum and weakly positive to the French serum. It would require further study.

The 12 check plants were definitely normal throughout and did not show yellow, brittle leaves. Four tests with antisera were lost, but eight were completed and were negative.

These experiments are considered as definitely showing that the disease of sugar beets in Oregon and the so-called "Salinas yellows" are in reality virus yellows as known in Europe, conforming in symptomatology, in mode of transmission, and in the serological reactions.

Numerous serial transfers of the virus yellows have been made using as virus sources the plants that showed virus yellows as a result of aphid transmissions. For example, Plant 1 of the experiment reported in Table 1 had aphids colonized a few days upon it. Then the aphids were transferred to healthy sugar beet seedlings and fed for two days before the test plants were sprayed with nicotine. Six out of the 10 inoculated plants showed virus yellows. In a companion experiment, 5 plants inoculated with virus tracing back to an earlier inoculation from a plant from the Jacobs field at Salinas, 3 out of 5 plants became diseased. Of the 10 checks which had similar treatment as to caging, etc. -- except no aphids were applied -- none became diseased.

Table 1. Results from inoculation test at Beltsville, Maryland, December 13, 1951, in which a "Salinas yellows" sugar beet from Jensen field and one from the Jacobs field, located near Salinas, were the respective sources of inoculum. Ten apterous aphids (Myzus persicae Sulz.) were placed on healthy 2-leaf stage sugar beet seedlings. Results were read January 31 and February 1, 1952, and re-checked February 15. Lower leaves becoming yellow and brittle were considered decisive of virus yellows.

Virus source: Plant from Jensen field				Virus source: Plant from Jacobs field				Check -- No aphids			
Test plant	Virus yellows symptoms	Date	Serum test	Test plant	Virus yellows symptoms	Date	Serum test	Test plant	Virus yellows symptoms	Date	Serum test
1	+	1/31/52	+	11	-	2/1/52	-	20	-	1/31/52	-
2	-	"	-	12	-	"	-	21	-	"	-
3	+	1/29/52	++	13	+	"	+	22	-	"	-
4	+	1/31/52	+	14	+	"	+	23	-	"	-
5	-	"	-	15	+	"	±	24	-	"	-
6	+	"	±	16	+	"	+	25	-	"	-
7	+	"	++	17	+	"	+	26	-	"	-
8	+	"	+	18	+	"	-	27	-	"	-
9	+	2/1/52	+	19	-	"	-	28	-	2/1/52	-
10	-	"	-					29	-	"	-
								30	-	"	-

Table 2. Results from inoculation test at Beltsville, Maryland, January 8, 1952, in which a "Salinas yellows" sugar beet from Salinas Valley Vegetable Growers Exchange field was the source of inoculum. Aphids (Myzus persicae Sulz.) were colonized one month on this plant, then transferred to test plants (2- to 4-leaf stage), about 25 aphids per plant. Feeding period was 2 days. When read March 21, 1952, lower leaves becoming yellow and brittle were considered decisive of virus yellows.

Test plant	Check -- No aphids: Virus yellows 2/21/52	Serological test: Date	Result	Test plant	Aphids from "Salinas yellows" sugar beet: Virus yellows 2/21/52	Serological test: Date	Result
1	-	3/21/52	lost	13	±	3/21/52	-; +
2	-	"	lost	14	+	"	+
3	-	"	lost	15	-	"	±
4	-	"	-	16	+	"	+
5	-	4/1/52	-	17	+	"	+
6	-	"	-	18	-	1/29/52; 4/1/52	-; -
7	-	"	lost	19	+	4/1/52	+++
8	-	4/2/52	-	20	+	"	+
9	-	"	-	21	+	"	+
10	-	4/1/52	-	22	-	4/2/52	-
11	-	4/3/52	-	23	+	"	++++
12	-	"	-	24	-	"	-
				25	-	"	-; -
				26	+	4/3/52	+++
				27	-	"	+ (weak)
				28	+	"	+++
				29	+	1/29/52; 4/3/52	+; +++
				30	+	" "	+; +++
				31	+	4/3/52	++
				32	-	"	-
				33	+	"	lost

DISCUSSION

Virus yellows, a serious disease of sugar beet and forage beets in Europe, is widespread in the United States. The first evidence available came from serological tests. The positive reactions of juices of suspected plants to rabbit antiserum of virus yellows made with European antigens, taking into account the known specificity of the reaction, convinced the writer that the disease was present in Michigan, Colorado, Utah, Oregon, and California, since plants exhibiting virus yellows symptoms collected at all these places repeatedly gave positive reactions. Other types of leaf yellowing did not give positive readings. Transmission by mechanical inoculations has been accomplished in two experiments with juices of yellowed sugar beets from Michigan and in another experiment with juice from a test plant infested with virus by means of aphids fed on a plant with "Salinas yellows". Transmission of virus yellows by means of aphids is, however, considered decisive. Abundant evidence of such transfer is presented, diseased plants from Oregon and from three separate fields in California being virus sources. Furthermore, the test plants with virus yellows have in turn served as virus sources in serial transfers. The inoculated sugar beet seedlings have been tested with antiserum of virus yellows, and the serological tests have confirmed the diagnosis based upon symptoms.

It seems probable that virus yellows has been present in the United States at least since 1940 when the writer first noted a condition of sugar beets in Colorado that he described at the time as brittle yellows. The early attempts at transmission by means of aphids were not successful, undoubtedly due to faulty technique[3]. The observations made in 1950, 1951 and 1952 indicate that the disease appears fairly late in the growing season in Michigan, Colorado, and Utah and may be localized around carry-over sources of infection. Thus, in Colorado and Utah, Dr. Hull noted the disease in proximity to sugar beet breeding stations and not in commercial fields distant from the stations. In California and Oregon, on the other hand, year-round activity of the aphid vectors, over-lapping of commercial sugar beet crops or of sugar beet seed crops, together with proximity of sugar beet fields to spinach or red garden beet fields, insure the continuity of the virus -- as opposed to the break in the virus cycle that winter weather brings about in all other sugar beet districts.

It is the opinion of the two European specialists who have seen the disease in the United States that the disease here is of mild type. The effects that the disease may have upon yield or quality of our sugar beet crops is not known. However, the presence of this potentially serious disease poses immediate problems for consideration by the industry, and the inauguration of appropriate research to find ways and means for its control.

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[3]Possibly because the aphids were fed on the youngest, instead of the older, yellowed leaves.

HOP-NETTLEHEAD, A VIRUS DISEASE NOT FOUND IN THE UNITED STATES

Donald P. Limber

The virus disease nettlehead (Chlorogenus humuli Holmes) is reported to cause serious losses in the hop yards in England and some parts of continental Europe. It has not been identified amoung the virus diseases of hops established in the United States. We do have at least one disease which causes somewhat similar symptoms, the trouble known as "slip-down" in the Pacific Northwest, but the differences between nettlehead and slip-down are sufficient that, unless they should eventually be shown to be due to climatic causes or to the reaction of different host varieties, we must consider the two diseases as distinct.

All persons interested in hop culture should be alert to detect any outbreak of nettlehead. Imported stocks are given particular attention by the Bureau of Entomology and Plant Quarantine through its postentry inspection. Information on this disease follows:

Other Names: Humulus virus 2, silly-hill disease, infectious sterility virus of hops.
Host: Humulus lupulus L.
Distribution: Czechoslovakia, England, Germany, Poland.
Symptoms: Infected plants do not climb well, and if severely affected they are bunched on the lower 3 to 5 feet of the support. There they present a rather dense mass of foliage owing to the excessive number of branches. The stems are spindly, stiff, and short. These symptoms are most obvious in cool weather and become masked as the temperature rises. Affected plants may thus show some recovery as the season progresses.

The leaves are smaller than on normal plants. The younger, smaller ones usually show upcurled margins. In the older leaves the upward curling disappears. There is a slight clearing of the veins, which is very evenly distributed when viewed in dry pressed specimens. The vein clearing is much less prominent and not so irregular as that seen in hop mosaic.

Production of cones is greatly reduced. Blattny and Vukolov (1) have stated that the only reliable external sign of the disease is the more or less complete sterility of the affected hop plants during three consecutive years.

The disease can be transmitted by grafting. Blattny and Vukolov have reported insect transmission, but as they have given a different vector in succeeding reports this probably needs confirmation. In the field it spreads to nearby plants, even to adjoining rows but not so readily as to plants in the same row. The variety Fuggles is particularly susceptible.

We are fortunate in being able to have this description reviewed by W. G. Keyworth, an authority on the nettlehead disease.

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BUREAU OF ENTOMOLOGY AND PLANT QUARANTINE, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE

TWO BIOLOGICAL FORMS OF PSEUDOPERONOSPORA CUBENSIS

Morris B. Hughes and Frank Van Haltern

Maintaining the downy mildew fungus of curcubits (Pseudoperonospora cubensis (Berk. & Curt.) Rostow.) on potted watermelon plants during the winter season is a standard practice in the cantaloupe breeding program at Experiment, Georgia. The failure at the Blackville, South Carolina Station to maintain this fungus on Congo watermelon despite the successful use of A and C cucumber for this purpose resulted in an exchange of mildew isolates between the authors to determine the cause of this difference in results.

Methods

Plants of A and C cucumber, Jumbo Hale cantaloupe, Congo and Georgia Wilt-Resistant watermelon were grown from identical seed lots at the two locations and tested for response to the two mildew isolates by spraying with spore suspensions when cotyledons had fully expanded but before the true leaves had emerged. Throughout the experiment the two mildew isolates were maintained on host plants in separate greenhouses at both locations so that mixture could not occur. No attempt was made to obtain like spore concentrations in the inoculum prepared from the two mildew isolates on any given day or for the same isolate for different days. The uniformity of results at the two locations and of different tests at the same location suggests that this failure to standardize did not adversely affect the results.

At Experiment, all material to be tested was grown in pots and kept in the greenhouse where the particular isolate used on it was maintained. At Blackville, a third greenhouse section was used to test the response of the hosts to the two isolates. This was necessary because, to reduce environmental effect, the plants at one end of a flat were inoculated with sporangia from one isolate while the plants at the opposite end of the flat were inoculated with sporangia from the other isolate. The central plants of each flat were left uninoculated. Wax paper was used to protect the remainder of the plants in the flat while the others were being inoculated.

The mildew isolate in use at the Georgia Station had been obtained from an old watermelon field in the Griffin area and will be referred to as the V isolate. The isolate supplied by the Blackville Station was obtained from cucumber plants growing in the greenhouse of the Charleston Truck Station and will be referred to as the H isolate.

Results

The results obtained at Experiment are presented in Table 1. Data are included on the preliminary testing of other cucurbits as well as on the four varieties tested jointly.

At Blackville only descriptive notes were taken on the three tests conducted there. The H isolate caused very severe and rapid damage to A and C cucumber and severe damage on Jumbo Hale cantaloupe, although symptoms were slower to develop than on the cucumber. On Congo and Georgia Wilt-Resistant watermelon symptoms were extremely mild, however, and consisted of a few small, definitely atypical, dark, non-sporulating lesions.

The V isolate was moderately severe on A and C cucumber and Jumbo Hale cantaloupe, but less so than H isolate, as shown both by time elapsing between inoculation and appearance of first symptoms and by time needed to complete destruction of the infected tissue. On both Congo and Georgia Wilt-Resistant watermelon, however, V isolate caused definite and considerable damage, the symptoms showing up as typical large irregular lesions.

Examination of the data in Table 1 shows general agreement with the results from Blackville. Although some Congo plants did suffer damage from the H isolate at Experiment, this isolate in general failed to cause appreciable damage on these watermelons. This difference in pathogenicity of these two isolates on watermelon is the principal basis for the conclusion that these isolates represent separate and distinct biologic forms of this species of fungus. The difference in intensity of reaction to these isolates of the cucumber and cantaloupe varieties tested jointly, even though pronounced in every test at both locations, is of lesser degree and is considered more in the nature of supplementary evidence for the above conclusion.

Further supporting evidence for this conclusion is found in the apparent difference in symptoms caused by the two isolates. The symptoms caused by the V isolate are first evident as yellowish blotches that spread widely in the leaf, usually for several days before the leaf tissue is killed or collapses. This appears to be true for watermelons, cantaloupes and cucumbers. Sometimes, especially when resistant cantaloupes are inoculated, the yellowing may take the form of

Table 1. The host reaction of seedling plants of several cucurbits to artificial inoculation with sporangia of two different isolates (V and H) of the fungus Pseudoperonospora cubensis (Experiment, Georgia).

Host variety	No. of plants	Date planted	Date inoc.	Mildew isolate	Progress of disease after inoculation, Number of days: 4	5	6	7	8	10	11	13	14	15	16	18
A & C cucumber		2-14	2-26	V			3-8*		4-10	7-10		10				
	10	2-19	3-3	V				1-5	9-10							
	13	3-24	4-7	V		4-5			5-10	7-10				9-10		
	11	3-26	4-9	V			5-10	9-10				10				
	12	2-14	2-29	H				4-9	6-9	10						
	8	2-19	3-3	H		1-3		10								
	15	3-24	4-7	H		4			10							
	13	3-26	4-9	H			5-10		8-10			10				
Jumbo Hale cantaloupe	12	2-14	2-26	V			1-5		2-9	2-10		4-10				
	22	2-19	3-3	V				1-4	3-9							
	14	2-14	2-29	H				1-10	3-10	9-10						
	19	2-19	3-3	H		1-3		10								
Freeman's cucumber	12	3-26	4-7	V		1			1-3	2-6				4-10		10
	11	3-26	4-7	H		1			2-5	5-10				10		
P.I. 179916 cantaloupe	11	3-26	4-7	V		1-2			3-7	5-10				9-10		10
	12	3-26	4-7	H		1-7			8-10	10						
P.I. 177334 cantaloupe	12	3-25	4-9	V			1		1-2			1-3				
	9	3-25	4-9	H			1-3		1-4			1-5				
Congo watermelon	11	2-14	2-26	V			0-1		0-9	1-10		4-10				
	7	2-19	3-7	V	4-10											
	8	2-14	2-29	H				0-1	0-1	0-4			0-4			
	8	2-19	3-3	H		0-1		1-8		1-9						
Ga. wilt-res. watermelon			2-26	V								6-10				
			3-3	V				1-2	3-10							
			2-29	H				0	0	0			0			
			3-3	H						0						
Cannon Ball watermelon	6	3-26	4-9	V			3-9		4-10			10				
	7	3-26	4-9	H					4-7			4-7				
Stone Mt. watermelon	11	3-25	4-7	V		1-6			8-10	10						
	11	3-25	4-7	H		1-6			3-9	3-10				3-10		
Imp. Kleckly watermelon	12	3-26	4-7	V		1-5			8-10	10						
	9	3-26	4-7	H		1-5			4-9	5-10				6-10		
Tom Watson watermelon	9	3-26	4-7	V		1-3			7-10	9-10				10		
	11	3-26	4-7	H		1-4			3-5	4-6				4-7		
Coles Allheart watermelon	7	3-25	4-7	V		1-8			7-10	9-10				10		
	8	3-25	4-7	H		1-4			3-7	3-7				3-8		
P.I. 169253 watermelon	11	3-25	4-7	V		1-3			5-10	5-10				10		
	12	3-25	4-7	H		1-3			3-8	3-8				3-10		
Yellow sum. straightneck squash	11	3-25	4-9	V			1-2		1-4			1-4				
	10	3-25	4-9	H			0-1		0-2			0-2				

*Zero -- no infection; 10 -- total infection or death. Data for each individual plant was not recorded. Paired figures denote the least and most severely infected plant, respectively.

threadlike extensions from the original point of infection, resulting in a zigzag or network pattern. The spore mass on the under side of the leaves appears grayish even when dense. The fungus fruits moderately well on all the varieties tested jointly.

In contrast, the symptoms of the H isolate first appear as tiny spots ringed by narrow yellowish bands. The spots are irregularly round and the tissue in the center collapses and dies quickly although the spots enlarge slowly. Sometimes the centers are dead before the visually infected area has spread more than a few millimeters. On A and C cucumber and Jumbo Hale cantaloupe the spots are very numerous. The sporulation on cucumber is extremely heavy, the spore mass appearing black to visual examination. On Jumbo Hale cantaloupe fruiting of the fungus is very sparse and on the watermelons extremely sparse, no fruiting at all having been seen at Blackville and only a trace around the narrow rims of lesions at Experiment.

The conclusion that distinct biological forms of this fungus occur is in keeping with the field observations of both Ellis (1) and Epps and Barnes (2) who suggested this possibility. The extent of diversity in this fungus awaits further investigation.

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CLEMSON COLLEGE EDISTO EXPERIMENT STATION, BLACKVILLE, SOUTH CAROLINA AND GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

NEW AND UNUSUAL RECORDS OF PLANT DISEASES

SOUTH AMERICAN LEAF BLIGHT ON HEVEA RUBBER IN HONDURAS

By B. H. Waite and V. C. Dunlap

South American leaf blight caused by Dothidella ulei P. Henn. was recently observed for the first time on rubbertree (Hevea brasiliensis (H. B. K.) Muell. Arg.) plantings of the United Fruit Company at Lancetilla and San Alejo. These plantings are 25 and 9 years of age respectively.

Although the disease had not previously been reported in the Republic of Honduras, its occurrence was to be expected since it has been established for some years in Costa Rica and Mexico and has more recently appeared in Guatemala. Spore dissemination has undoubtedly been accomplished by wind currents.

Typical symptoms of blight were observed on young and older leaves, leaf petioles, twigs, and green fruits. Considerable defoliation was evident. Although the conidial stage was found to be dominant, the pycnidial and ascigerous stages were also observed. Blight-resistant clones appeared immune to attack or were only mildly blighted.

The appearance of Dothidella ulei in Honduras and Guatemala apparently completes its distribution throughout all areas in Central America where rubber is grown commercially.

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A NEW PERUVIAN HOST FOR PHYTOPHTHORA CINNAMOMI

By C. Bazán de Segura

Phytophthora cinnamomi was reported (PDR 35: 335) last year as causing the death of more than 50,000 avocado trees in the Chanchamayo Valley of Peru. The same fungus was also isolated from rootlets of diseased papaya plants (Carica papaya). From the same area this fungus has now been isolated from rootlets of diseased plants of "roble blanco", Octea architectorum, likewise a member of the Lauraceae as is the avocado.

CENTRO NACIONAL DE INVESTIGACION AGRICOLA DE LA MOLINA

CHARCOAL ROT ON FLUE-CURED TOBACCO IN VIRGINIA

By Wilbert A. Jenkins

Charcoal rot, Macrophomina phaseoli, (Sclerotium bataticola) is extremely prevalent during the current season and is consistently associated with basal (shank) rotting of tobacco plants injured by the application of mineral oils for sucker control. No sporulation has been observed on field material but the bark and wood are peppered with imbedded sclerotia. Considerable pith and wood decay has been observed, resulting in lodging and death of plants, and there is little doubt that M. phaseoli accomplishes this once it has gained entrance into the plant under conditions of favorable temperature.

Early observations on material being sent in for diagnosis were not conclusive as regards etiology and development of the disorder. However, with several study periods in the field it became evident that the initiating factor was scald induced by the oil treatment. Rather severe burning of the bark at the base of plants was a common symptom but a truly amazing incidence of charcoal rot was found following penetration of the oil into the pith at the base of the stem. It could not be determined in most cases whether the application of oil had killed the seedling leaves or whether these had perished before the oil was applied, but invariably the petioles of such leaves were found to be functioning as blotters or wicks and thus conducting the oil into the pith through the leaf traces. Such petioles could be removed manually and on so doing the leaf trace was removed as a plug, thus exposing the entire leaf gap. Frequently, axillary buds which would have produced suckers, that had been killed by the oil treatment were also found to be functioning as wicks and conducting oil into the pith of the upper stem. With localized coincidence of high temperature and humidity, such injury predisposed soft rotting (Bacillus aroideae), but the prevailing drought of the current season made such conditions infrequent. Now that we have had rain, soft rot is again coming into the picture.

The amazing consistency of charcoal rot in the basal portion of plants injured by oil during the current season has raised several questions. Granting that M. phaseoli is a universal soil inhabitant, thus explaining its entry exclusively near the soil line, much uncertainty still exists regarding practical exclusion of several other more or less universal soil inhabiting fungi (as Rhizoctonia spp. and Fusarium spp., in particular) from the picture. Several possible explanations occur to the writer but in the absence of experimental evidence it would seem that 1) M. phaseoli is better adapted to drought conditions or 2) M. phaseoli succeeds relatively better on an "oil diet".

Comparable injury, often associated with excessive crop losses, are being observed this season from the usage of either oil emulsions or heavy grade, purified, mineral oil and with dosages of either varying from one-half to one teaspoonful of oil or oil emulsion per plant.

VIRGINIA AGRICULTURAL EXPERIMENT STATION, CHATHAM, VIRGINIA

CHARCOAL ROT OF IRISH POTATO IN TENNESSEE

By E. L. Felix

Charcoal rot, caused by Macrophomina phaseoli, resulted in 33 to 50 percent loss of Irish Cobbler and Kennebec potato tubers in home gardens in the Knoxville area at the time of digging in early July. The potatoes were left in the very hot, dry soil for 2 weeks or more after the vines died, despite some very early planting. The vines were healthy according to the owners. However, sclerotia of the fungus were found on the root of one dead plant. The black, mostly soft, rot occurred equally anywhere on the tubers and because of secondary invasion by the bacterial soft rot organism, Erwinia carotovora, late symptoms in most instances very closely resembled those of blackleg. No primary invasion by the bacterial soft rot organism was observed. Macrophomina phaseoli was isolated readily from the rotten tubers and on artificial inoculation caused infection of wounded healthy tubers within 14 hours and extensive rot in 3 days. Charcoal rot is moderately firm in the absence of secondary rots. Symptoms of induced rot suggest that blackening around lenticels does not necessarily indicate entrance through the lenticels. In culture, the tiny sclerotia were very numerous and uniform in size in the 41 isolates made, but no pycnidia have formed, so far [August 8].

No such serious rot of this type has ever before been observed by these home gardeners and the influence of the prevailing abnormally high temperatures and prolonged stay in the hot soil becomes apparent. There is some reason, however, to believe that more or less of this rot has occurred in Tennessee in other years, according to J. J. Bird and W. W. Stanley.

The average maximum air temperature in Knoxville for the 37 days (June 1 - July 7), preceding some harvests with heavy loss, was 92.2° F., which is slightly above the reported optimum for the growth of the fungus (minimum 46° F., optimum 88°, and maximum 108°). The official average temperature for June was 81.4° F., which was 6.7° above normal; the rainfall 1.98 inches, which was 2.12 inches under normal for the month. Scattered rainfall of 2.72 or less inches occurred over the area during the first week in July.

No reports on potato rots in the commercial areas of Tennessee have been received, possibly due to early harvest. However, the disease was found on the Knoxville market in potatoes grown on the Cumberland Plateau. Charcoal rot also was found by Miss Ellnora Butler on a Bliss Triumph potato, purchased locally on the market in May, but grown in Alabama.

Potato growers, especially those in the more northernly regions, should guard against charcoal rot this season, particularly if the widespread, abnormally hot weather continues and the disease should develop farther north than usual. Potatoes should not be left in the ground under conditions of very high temperature.

UNIVERSITY OF TENNESSEE AGRICULTURAL EXPERIMENT STATION, KNOXVILLE

THE NEW FRONTIER FOR PLANT PATHOLOGY

William C. Paddock

In 1948 shortly before his death, the eminent mycologist, H. M. Fitzpatrick was standing in a plant pathology laboratory. His eye fell on a dusty, cobwebby basket which had been used by Prof. H. H. Whetzel, one of the first American professors of Plant Pathology, on his mycological forays. The basket was resting under a table supporting a Warburg apparatus which was chugging at full tilt on an experiment to determine the mode of action of copper on fungus spores. Dr. Fitzpatrick remarked that there was the difference between plant pathology when he was a graduate student and plant pathology when this writer was a graduate student. The difference between the mycological collection basket and the Warburg.

Like every science, plant pathology has changed, and changed radically during the past forty years. It would be regrettable if it hadn't. But there is, perhaps, another change at hand for it almost as great.

Such enormous progress has been made in the field of plant pathology, in selection of newer and better fungicides, in the juggling of plant genes for disease resistance that the results are truly spectacular. Modern fungicides now have such over-all good qualities that consideration of a cheaper, much less effective fungicide is nearly unthinkable. The progress already attained in disease resistance, while admittedly only a beginning, makes the plant breeder work in steps of increments that lack the seven-league-boot strides of the first breeders.

This is to be expected. Millardet radically increased yields with his Bordeaux. Is it to be expected that the compounding of a new fungicide today will give yields as great over what Bordeaux gives as the increased yields that Millardet gave the growers when they knew no fungicide? Or will the flax breeder come up tomarrow with a resistant variety which so far out yields todays varieties, that Bolley's increased yields at the turn of the century are made to look small? It's possible, yes. But the increases that the new fungicides and the new resistant varieties give us today are usually much less. They are frequently increases of only a few bushels or pounds more to the acre.

That this is not a worthwhile aim would be stupid to infer. However, because this is true, because he thinks in a few pounds or bushels, is it possible that the plant pathologist may have settled into a manner of thinking which can cloud his view of what his science might do to solve the basic world problem today?

Nearly every newspaper and magazine today carries some article on the world food situation as correlated with population growths. We hear so frequently that two-thirds of the world is under fed (sometimes it is three-fourths, but why quibble) that it no longer arrests our eye as it scans the page. Each day, we are told, there are 60,000 (some say 70,000) more new mouths on this earth that must be fed -- adding further to the strain of the undernourished economies of the world.

But that most of the world is undernourished and that these undernourished countries are underdeveloped countries should stir a plant pathologist every time he hears or sees these facts. The underdeveloped countries need the pathologist, in a way they need no other specialist. If a pathologist projects himself with his present knowledge back to the early days of Prof. Whetzel and visualizes the advances he could have made in this country at that time, then he can see why these countries must have his help. Let not the pathologist think in terms of the two or five percent increases that a new fungicide or disease resistant plant may give us in this country, but think in terms of twenty, fifty or even three hundred percent increases in the underdeveloped country. Such increases are possible.

No other science offers the hope for increases in a backward country that plant pathology does when coupled with the science of the plant breeder. There are, of course, any number of additional ways to increase yield -- machinery, fertilizer, crop mangement, insect control and resistance -- besides disease control. But fertilizers and machinery are expensive, and insect resistance difficult to breed for when compared to disease resistance. Crop mangement changes come hard to a people who have not changed their methods in a thousand years.

Plant diseases these countries all have -- many of the countries are tropical and diseases are rampant. Hand dusters are cheap and fungicides cheaper but, even so, they may be too expensive for the primitive farmer, even if he had the training, to use them. But disease resistant varieties, if available, are a different matter. The seed can be produced so that it would be as inexpensive as the seed the farmer now uses. It would help if he could be made to change his cultural practices, but if he didn't, he would still have increased yields.

It is not by coincidence that the remarkable achievements of the Rockefeller Foundation in Mexico, under the directorship of Dr. J. G. Harrar, former head of plant pathology at Washington

State, have been accomplished with a staff that has contained more plant pathologists than any other plant science speciality. This group is revolutionizing the agriculture of Mexico by making that country self-sufficient in corn for the first time in recent history -- in spite of the largest population in Mexican history. The same story is being repeated in wheat. Nor is it a coincidence that a much smaller organization in Guatemala, established by Iowa State College, is approaching in Guatemala the same goals reached in Mexico, under the guidance and almost single handed efforts of Dr. I. E. Melhus -- another plant pathologist.

The opportunity to repeat these advances in most of the undeveloped areas of the world now exists under our Government's Point IV program as executed by the Technical Cooperation Administration which is now undergoing a tremendous expansion. The Point IV program is not an ephemeral thing that is going to disappear within a couple of years. On the success of Point IV rests our own standard of living. With the world, excepting ourselves and a few other countries like Canada and New Zealand, suffering from malnutrition, we cannot otherwise maintain our living standard. No one minimizes its significance. To paraphrase President Truman, "who can say but that this will be our most important undertaking." Point IV demands on plant pathology will increase in the future.

While it is up to the government to present the world importance of Point IV dramatically to both the scientist and non-scientist of our country, the responsibility for education in the program's need does not end here. If newly trained plant pathologists are not made to see the need for their science in the underdeveloped countries, if the graduate programs do not awaken in them the potentials of their science in such a country, the government will not be able to find the pathologists it must have for its program. The responsibility for the participation of plant pathologists in the Point IV program rests with the men training graduate students in that field today. If this science is to play the role it must, a segment of the training now being given to graduate students in plant pathology must be directed towards this role.

The manner of training our pathologists in light of this would vary from school to school. This writer does not visualize the need for a separate course (which a student could or could not choose to elect) covering the method of approaching and handling a pathological problem in an underdeveloped area. Instead the material should be inserted, wherever opportune, in any or all courses now given.

Thus, for example, when the instructor discusses the properties of a good fungicide, let him give them for a grower in his State but continue the discussion to include what the properties would be for an underdeveloped country -- they, would, of course, be much different. The ideal fungicide for a poverty stricken agricultural system must be infinitely inexpensive. Furthermore, farmers who have never used a fungicide and who can be given little instruction and supervision in their use must have an infinitely safe fungicide.

When the instructor discusses in his course the degree of disease control practical, let him cover the relationship between cost of control and return, the emphasis on quality demanded on our market today and the cost of such quality. Contrasting this with the practical control in an agriculturally backward country where the emphasis would be first and foremost on quantity not quality. The degree and type of disease control necessary would thus be different.

In discussions on breeding for disease resistance, the contrast between what is required in our own agricultural system and what is needed in a phytopathologically undeveloped country such as Afghanistan could be illustrated. With no resistant varieties, such a country could use a highly rust resistant wheat, but it could better use a wheat with moderate rust, smut and scab resistance -- a wheat probably quicker to develop. Stress would be placed on the speed and success of improving a variety by mass selection in contrast with a true breeding program.

And when discussing the importance of plant pathology to our own agriculture today, let the student be told of the remarkable changes the science, with its present degree of development, can produce in a nation now lacking the science. Let the student never forget the magnitude of the advances if one, with present day knowledge, could have tackled the problems of Berkeley's day. For like the "Triton among the minnows" his power to increase production in an underdeveloped area might dwarf the increases he could perform in our own highly technological agriculture. The student should know the revolution possible in a backward country when crop production is boosted a mere 5 percent. It should be stressed that while the plant pathologist can produce virtually unlimited changes for good in such a country, that these changes will directly effect every American's living standard -- the moral purpose of each State Agricultural College where he is most likely to be employed if he remains in this country.

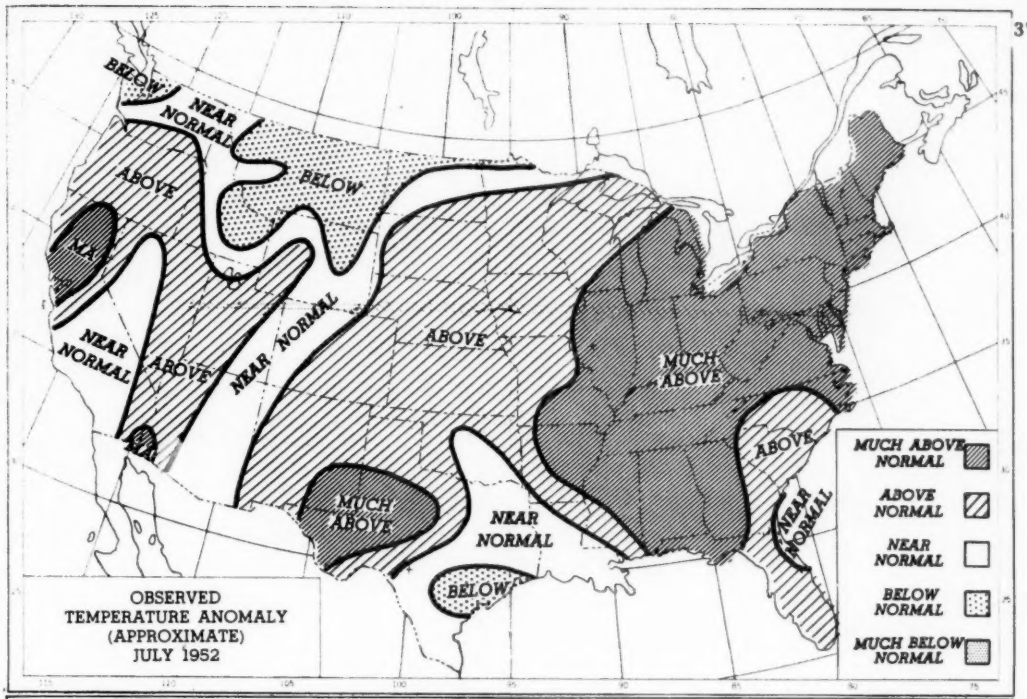
But there will be other direct advantages to our country and our agriculture from plant pathological work in the underdeveloped countries of which our graduate students should be made aware. Cases in point are numerous. The remarkable development in Mexico by Dr. Norman E. Borlaug of wheat resistant to the now threatening race 15 B of stem rust might well be the basis of

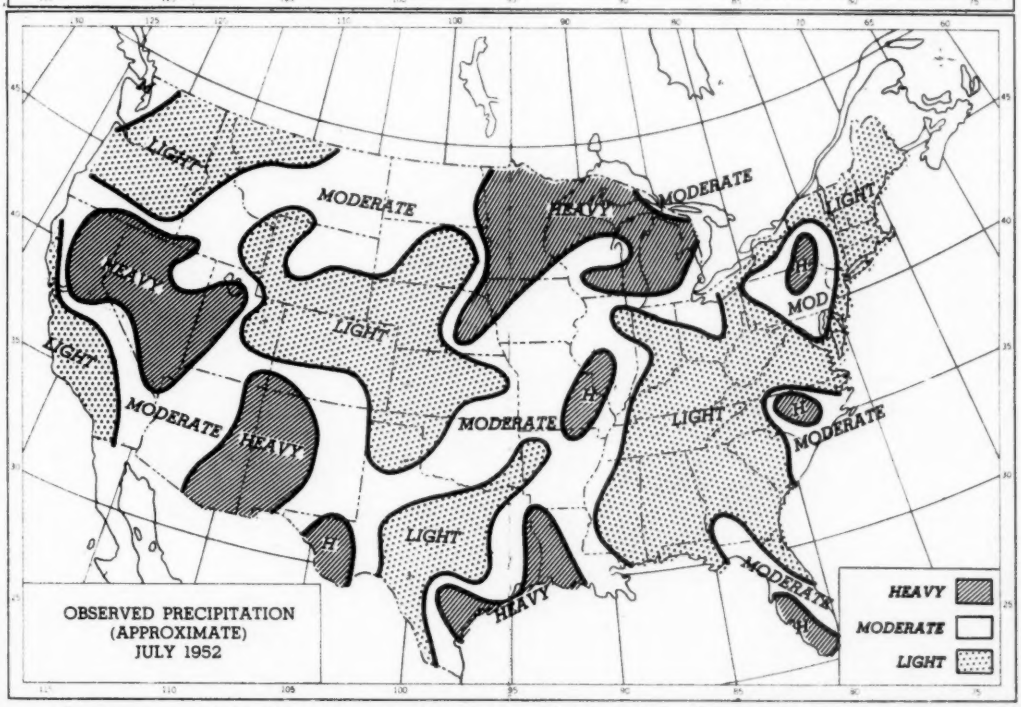
survival of our wheat crop in a few years. Recently it has been brought to the writers attention, by a pathologist just returned from Indonesia, that two disastrously virulent Sclerospora diseases of corn occur in that country. That these two diseases can destroy with ease every U. S. corn variety so far tested is interesting enough, but more interesting is the ignorance of our corn researchers in this country, a lack of interest to the extent that these diseases would never have been considered it it were not for a pathologist working in Java under Point IV. The world has many diseases whose potentialities in this country are unknown. We must know what these potentialities are in the event that they be introduced here either accidentally or through the encouragement of an agressor nation.

The incorporation of this role of plant pathology into the present method of teaching plant pathology might well produce more men than we now have willing to accept the challenge of this new frontier for plant pathology -- plant diseases in the underdeveloped country. Presenting plant pathology as an active combatant of world communism might be as radically different from the present approach as the mycological collection basket and the Warburg apparatus. One cannot minimize the importance of a science which seeks to destroy that on which communism feeds -- hunger.

Thus, plant pathology teachers must devote a portion of their energies to training men for this new frontier in plant pathology, for the role the science has in leading the underdeveloped countries to produce the food they must have, as well as for the role of crop protection in this country through disease investigation abroad. If pathologists are unwilling to carry their science to the underdeveloped, undernourished nations of this world, must not the blame rest partly on the departments training these men?

PENNSYLVANIA STATE COLLEGE, STATE COLLEGE, PENNSYLVANIA





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.